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The Diagnostic Pitfalls of Subarachnoid Hemorrhage from Intracranial Aneurysms

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The subtle manner in which subarachnoid hemorrhage frequently presents may delay appropriate treatment. The patient may deteriorate and die from aneurysmal rebleeding or from cerebral ischemia associated with vasospasm before the true nature of the disease is recognized. Five patients are described in whom subarachnoid hemorrhage was initially not recognized. Pitfalls in diagnosis are discussed, and an outline is presented for the evaluation of patients suspected of harboring ruptured intracranial aneurysms.

THE OUTLOOK for patients with ruptured intracranial aneurysms is improving. Yasargil, using microsurgical techniques, has achieved the remarkably low operative mortality of 1.9 percent in the 373 patients he operated upon from January 1970 through July 1974.[1] The use of antifibrinolytic agents is being investigated.[2,3] These agents inhibit clot lysis, and theoretically encourage the formation and preservation of a firm blood clot within and around a ruptured aneurysm, thus reducing the risk of rebleeding. This may prove to be a valuable form of therapy, used either alone or as an adjunct to surgical obliteration of the aneurysm.

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Slosberg champions an aggressive nonsurgical hypotensive regimen in the treatment of intracranial aneurysms.[4] Though many neurosurgeons are skeptical of the safety of Slosberg's approach, his work has certainly emphasized the importance of careful monitoring of blood pressure and control of hypertension in the acute and chronic management of the patient who harbors an intracranial aneurysm.

Stereotactic methods under development allow the obliteration of aneurysms without open craniotomy,[5,6] and Serbinenko has been successful in catheterizing specific intracranial arteries selectively.[7] Refinement of his methods may make possible the obliteration of aneurysms with little more risk than that of cerebral angiography.

As the treatment of ruptured intracranial aneurysms improves, it becomes more and more im-

portant to institute appropriate therapy as soon as possible, but it is surprising how often subarachnoid hemorrhage is misdiagnosed.[8] Inappropriate measures which may actually harm the patient are instituted, and appropriate measures are inadvertently withheld. It is relatively easy to recognize subarachnoid hemorrhage in a young or middle-aged patient presenting with a history of sudden headache followed by stiff neck, lethargy, hemiparesis or seizures. However, subarachnoid hemorrhage frequently presents in a more subtle form. It is exceedingly important that these subtle variations be recognized and therapy instituted before the patient deteriorates from rebleeding or cerebral ischemia associated with vasospasm.

This author has seen a number of patients with subarachnoid hemorrhage and in whom clinical features were at first confusing. The following patients are examples. They were all first seen by well trained, experienced, conscientious physicians. The diagnostic pitfalls these doctors encountered illustrate that in order to avoid misdiagnosing subarachnoid hemorrhage, a physician's index of suspicion must frequently be very high.

Reports of Cases

CASE 1. A 50-year-old woman reported that an "exploding" headache had suddenly developed one evening after she ate a lobster dinner. The patient induced vomiting to relieve severe nausea which developed immediately following the onset of the headache. In a hospital emergency room, a history of migraine headaches was given, though there had been no significant headaches during the five years preceding the present illness. There was no history of hypertension. The patient was neurologically normal. It is not known if there was nuchal rigidity. The patient was told that blood pressure was elevated, and a diagnosis of hypertensive headache was made. Consideration was also given to a diagnosis of acute gastroenteritis secondary to the ingestion of contaminated shellfish. Two injections (presumably of antihypertensive medication) were given and the patient was sent home with an antiemetic. The symptoms persisted and four days later an internist was consulted and he admitted the patient to hospital. At that time, there was nuchal rigidity and the patient was somewhat lethargic but oriented. Deep tendon reflexes were hyperactive on the left. Blood pressure varied between 140/80 and 170/90 mm of mercury.

On lumbar puncture, bloody cerebrospinal fluid was found with a supernatant that was notably xanthochromic. On transfemoral catheter cerebral angiography, a right internal carotid artery aneurysm was noted at the origin of the posterior communicating artery (Figure 1, left). There also was a small (3 mm) aneurysm on the trunk of the left middle cerebral artery (Figure 1, right). The patient at first declined surgical operation, but later consented, and the right internal carotid

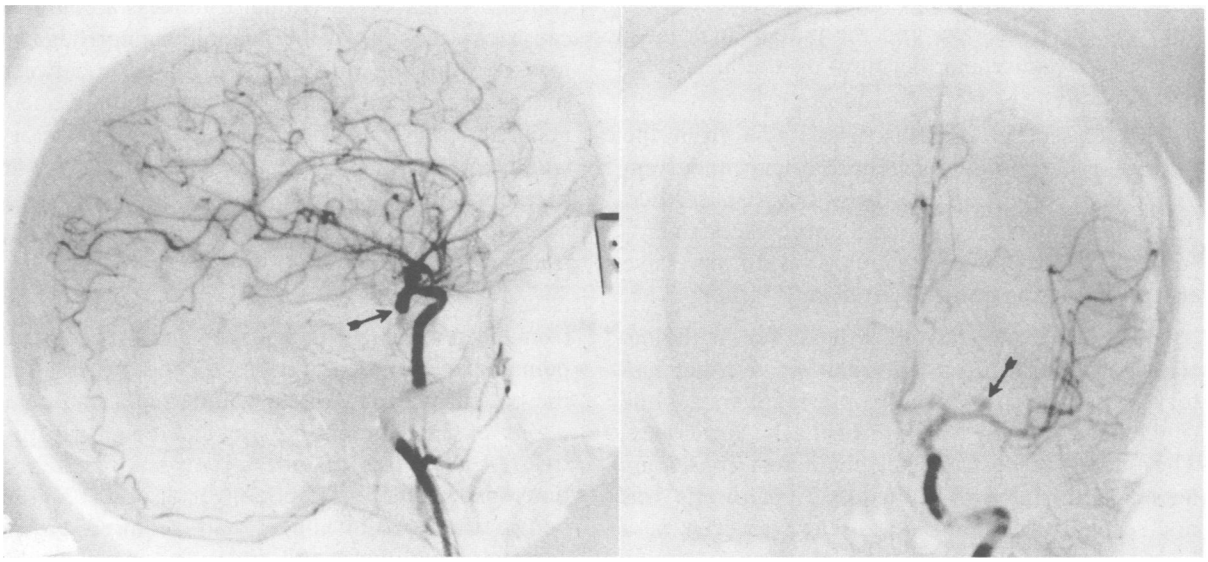

Figure 1.—(Case 1) In a 50-year-old woman, a right posterior communicating artery aneurysm (arrow, **left**) which had ruptured was noted on angiography. This lesion was successfully obliterated, but an aneurysm of the left middle cerebral artery (arrow, **right**) remains.

artery aneurysm was obliterated by an intracranial microsurgical approach 11 days after the subarachnoid hemorrhage.

The patient recovered without neurological deficit and returned to work. Over the four years since the procedure, the patient has continued to do well, though antihypertensive medication has been required for persistently elevated blood pressure. A mild seizure disorder may be present and diphenylhydantoin is being taken. So far, the patient has declined the recommendation that repeat angiography be done to rule out progressive enlargement of the left middle cerebral artery aneurysm.

Comment

When this patient presented to a hospital emergency room immediately following subarachnoid hemorrhage, a diagnosis of hypertensive headache was made because the blood pressure was elevated. However, elevation of the blood pressure is common following subarachnoid hemorrhage. Even if a patient has a history of hypertension, the development of headache cannot be ascribed with certainty to the hypertension alone, and the physician must keep in mind the increased incidence of subarachnoid hemorrhage in hypertensive patients.

It is not uncommon for subarachnoid hemorrhage to be misdiagnosed as some sort of abdominal disorder (see also Cases 3 and 5). The nausea and vomiting which frequently accompany subarachnoid hemorrhage may mask the importance of the headache, and the patient may have abdominal tenderness because of the persistent vomiting.

This patient is at considerable risk from the remaining left middle cerebral artery aneurysm and the hypertension increases this risk further. This author would recommend prophylactic microsurgical obliteration of the aneurysm if progressive enlargement was seen on serial angiography.

CASE 2. A 41-year-old woman fell at home, striking her head on a television set. Further history, obtained subsequently, indicated that while watching television a severe headache developed. The patient arose from her chair, lost consciousness and then fell. This sequence of events was not recognized when the patient was seen in a hospital emergency room the morning following the event. Since ecchymosis was present around the left eye, it was assumed that the headache was the result of the head trauma. The patient was neurologically normal. It is not known whether or not nuchal rigidity was present at that time. The patient was observed in the emergency room for several hours, then released. However, the symptoms persisted, and an internist was seen two days later. The patient was admitted to hospital, at which time the blood pressure was 120/60 mm of mercury. The orbital ecchymosis was again noted. Results of funduscopic and neurological examinations were normal except for a suggestion of impaired graphesthesia in the left upper extremity. Nuchal rigidity was present.

Delayed venous drainage of the right cerebral hemisphere was noted on brain scan, though findings on blood brain barrier scintiphotos were normal. A lumbar puncture produced bloody cerebrospinal fluid with a pressure of 270 mm. The supernatant was moderately xanthochromic. On transfemoral cerebral angiography, an aneurysm of the right middle cerebral artery was seen. Craniotomy was carried out four days after the subarachnoid hemorrhage and the aneurysm was obliterated microsurgically. The patient did well postoperatively, though a minor seizure disorder subsequently developed and administration of anticonvulsants was required.

Comment

The subarachnoid hemorrhage caused the patient to lose consciousness and fall. The case was originally misinterpreted as one of straightforward cranial trauma. Since cranial trauma is a common cause of secondary subarachnoid hemorrhage, it may at times be difficult to distinguish between subarachnoid hemorrhage due to a ruptured aneurysm and that due to trauma. A careful history will help, but angiography may be necessary to investigate the possibility that a ruptured aneurysm led to the event responsible for the cranial trauma.

CASE 3. A 55-year-old man was awakened from sleep one night by headache and severe neck pain. When admitted to hospital shortly thereafter, the patient was nauseated, vomited and had complaint of anterior chest pain and epigastric distress. A diagnosis of anterior myocardial infarction was considered. Peptic ulcer disease 12 years before the present illness had required gastric surgical operation, and recrudescence of this problem was also considered as a diagnostic possibility. Morphine was given and the patient soon

became lethargic to a degree out of proportion to the dose administered. Pronounced confusion was also noted, and the possibility of excessive self-medication at home was considered, as was the possibility of a psychotic reaction of some sort. Blood pressure was 120/60 mm of mercury. Findings on funduscopic and neurologic examinations were normal except for the lethargy and confusion.

It is not known if objective nuchal rigidity was present at the time of admission to hospital, but three days later an orthopedic consultant, called because of the patient's continuing neck pain, noted a stiff neck. A diagnosis of acute cervical strain was made, and the patient was placed in cervical traction.

Five days after admission neurological consultation was obtained and bloody cerebrospinal fluid with xanthochromic supernatant was found on lumbar puncture. Transfemoral cerebral angiography was carried out with the left vertebral and both carotid arteries being injected. The source of the subarachnoid hemorrhage could not be found, though in retrospect mild vasospasm was present in the left internal carotid artery (Figure 2, left). The lethargy and confusion resolved and the patient was discharged from the hospital 11 days following the subarachnoid hemorrhage.

One month after the initial hemorrhage, there was recurrence of severe headache and effective vision in the left eye was lost. Upon admission to hospital a large preretinal hemorrhage covering the left macula was noted. There were preretinal hemorrhages in the right fundus as well. Blood pressure was 110/70 mm of mercury, and nuchal rigidity was present. The patient was lethargic, but otherwise neurologically intact.

On lumbar puncture five days after the recurrence of the headache, xanthochromic cerebrospinal fluid was noted. A large anterior communicating artery aneurysm (Figure 2, right) was seen on cerebral angiography. The patient initially refused surgical operation, and 21 days following the recurrence of headache, still another sudden headache developed with nuchal rigidity. Lumbar puncture was not felt to be necessary. The patient agreed to surgical operation, and five days later craniotomy was carried out and the aneurysm obliterated with microsurgical techniques. A low-grade wound infection from the Providence group of Gram-negative organisms developed, but this responded well to gentamicin. The patient did well and returned to work. Vision in the left eye subsequently improved.

Comment

The presentation of the initial subarachnoid hemorrhage in this patient was confusing. Several diagnoses were initially considered, including anterior myocardial infarction, recrudescence of peptic ulcer disease, drug overdose, acute psychotic reaction and cervical strain.

The large anterior communicating artery aneurysm did not fill at the time of initial cerebral angiography, though in retrospect there was internal carotid arterial vasospasm which was a clue to the presence of the lesion. The aneurysm

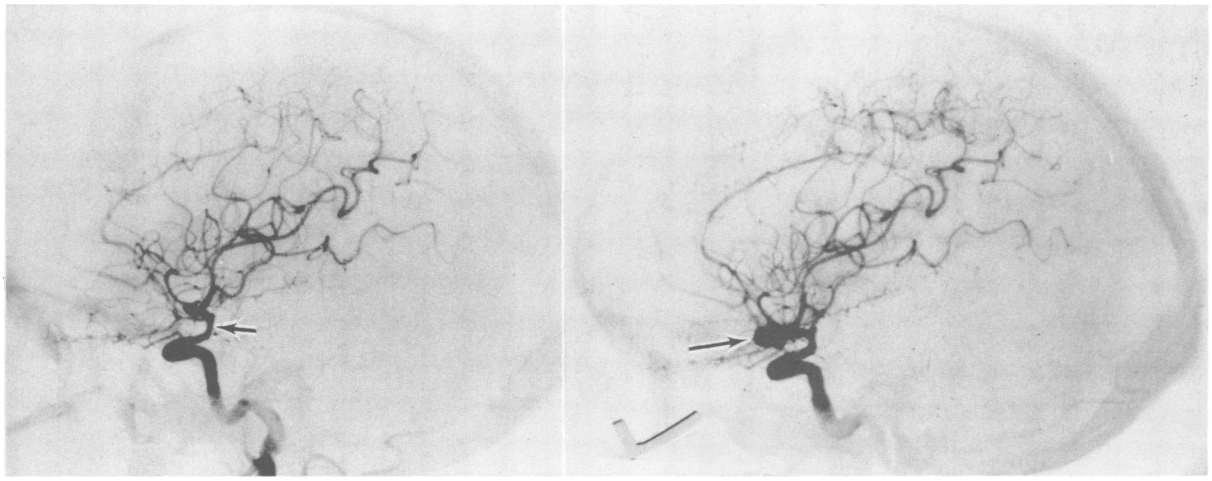

Figure 2.—(Case 3) In a 55-year-old man, a left cerebral angiogram (above, **left**) showed no aneurysm, though there was vasospasm of the left internal carotid artery (arrow). Left cerebral angiogram one month later (above, **right**) showed a large anterior communicating artery aneurysm (arrow).

filled well on a subsequent angiogram. This case well demonstrates the point that cerebral angiography should be repeated if the initial study does not show the source of an unexplained subarachnoid hemorrhage. The possibility that the source of the hemorrhage may be occult spinal pathology should be kept in mind. Intracranial subarachnoid hemorrhage can be mimicked by hemorrhage from a spinal cord vascular malformation, especially if the lesion lies in the cervical spinal cord. Intradural spinal tumors, particularly ependymomas, can also present initially with subarachnoid hemorrhage. If the source of the hemorrhage cannot be shown by cerebral angiography, then myelography and spinal angiography should be considered.

CASE 4. In a 40-year-old woman, a stiff neck and occipital headache suddenly developed while she was playing tennis. An orthopedist, who was consulted, found no neurological abnormality. It is not known whether or not objective nuchal rigidity was present at that time. The headache persisted for two weeks, then subsided. One month later, again while she was playing tennis, severe headache developed and the patient temporarily lost consciousness. Upon admission to hospital, blood pressure was 140/80 mm of mercury. The patient was awake though confused, but shortly thereafter suddenly lost consciousness again and became apneic. After intubation, the patient regained consciousness but was combative. Right hemiparesis was present. The blood pressure then was 100/60 mm of mercury. On funduscopic examination, preretinal hemorrhages in the left eye were noted. Lumbar puncture was not done, since the diagnosis of subarachnoid hemorrhage seemed secure without it.

Two days after admission, a left infraclinoid carotid aneurysm (Figure 3) was noted on transfemoral cerebral angiography. The patient became fully awake and the right hemiparesis resolved, though there was some remaining confusion and a memory deficit. Five days after admission, a left frontotemporal craniotomy was carried out and the aneurysm successfully obliterated.

Following the craniotomy the patient was neurologically intact and mental status continued to improve until the fifth postoperative day when lethargy and aphasia with hemiparesis on the right developed. Cerebral angiography was repeated and severe spasm of the internal carotid, middle cerebral and anterior cerebral arteries was noted. Intravenously administered phenylephrine was used to increase the blood pressure. When the blood pressure dropped below 120 systolic, the patient became totally unresponsive and decerebrate posturing of all extremities developed. Gradually tolerance to the phenylephrine developed. Levarterenol was substituted, but the blood pressure become unresponsive to that medication as well.

On a third cerebral angiogram three days after

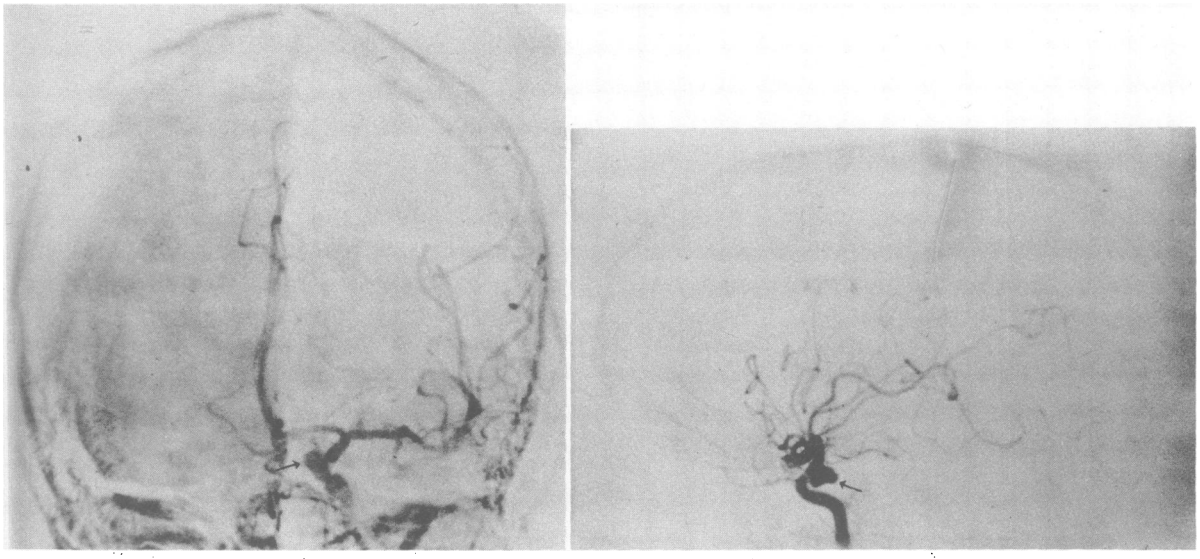

Figure 3.—(Case 4) In a 40-year-old woman, the infraclinoid internal carotid aneurysm (arrows) was clipped five days after the subarachnoid hemorrhage.

the onset of neurological deterioration, further increase in the spasm was noted. A left superficial temporal-middle cerebral artery microsurgical bypass was done, in the hope that this procedure would increase cerebral perfusion enough to reverse the deterioration. Immediately following the procedure the blood pressure was allowed to drift toward lower levels. Decerebrate posturing was no longer present. The next morning the vasopressors were discontinued, and the systolic blood pressure stabilized at 90. The neurological status began to improve, and 72 hours after the microvascular procedure the patient was fully responsive, was taking oral fluids and had returning function of the right arm. Neurological improvement continued, and at present—one year and a half after the episode—speech is normal and there is full use of all extremities. The patient is completely independent in the activities of daily living, but still lacks initiative and has some deficit in recent memory. As improvement continued, the superficial temporal arterial pulse gradually diminished, finally becoming imperceptible. On cerebral angiography two months after the microvascular procedure, it was noted that the microvascular bypass was no longer patent. Presumably it closed as normal cerebral circulation became reestablished.

Comment

In this patient, there almost certainly was minor subarachnoid hemorrhage one month before the major hemorrhagic episode—reflected by the sudden development of a stiff neck and occipital headache while playing tennis. Hemorrhage presenting in this form is extremely subtle and confusing, but the severity and persistence of the headache and the absence of a specific precipitating traumatic event might have been clues that the symptoms represented more than a simple cervical strain.

The major hemorrhage which precipitated admission to hospital almost resulted in immediate death. The patient did well immediately following the craniotomy, only to be placed at great risk again by the development of vasospasm associated with severe cerebral ischemia. The author feels that cerebral vasospasm in this patient represents the delayed phase of vasospasm which develops in approximately two thirds of patients following subarachnoid hemorrhage with or without intervening craniotomy.[9-12] (See also Case 5.) In humans, delayed vasospasm becomes maximal approximately one week following hemorrhage.[9] Delayed vasospasm is in some cases associated with severe cerebral ischemia leading to infarction,[13] and accounts for a number of the deaths resulting from subarachnoid hemorrhage. This type of vasospasm has proven resistant to direct treatment by any vasodilating drug now available, but if the ruptured aneurysm has been obliterated before the patient develops cerebral ischemia, then the ischemia can in some cases be reversed by the use of induced hypertension.[14,15] Induced hypertension presumably increases perfusion of the compromised cerebral tissue by taking advantage of the fact that circulatory autoregulation is impaired in the ischemic regions. Perfusion in these areas is directly proportional to the blood pressure. However, induced hypertension cannot be used safely if the patient still harbors a patent aneurysm, as Allcock and Drake have emphasized.[16]

The author has successfully used a regimen of induced hypertension in two other aneurysm patients severely threatened by cerebral ischemia which developed several days after craniotomy. In the patient reported above (Case 4), however, induced hypertension alone did not reverse the deterioration, and a microvascular shunt was carried out in an attempt to increase cerebral perfusion. It cannot be determined with certainty whether or not the microvascular bypass contributed to the patient's survival and neurological improvement, but the postoperative course suggests that it did.

Early diagnosis of a ruptured aneurysm and prompt obliteration of the aneurysm allow vigorous treatment of cerebral ischemia should it occur several days after the hemorrhage in association with delayed vasospasm.

CASE 5. A 37-year-old woman awakened early one morning with a severe headache. Nausea, vomiting and diarrhea followed shortly, and the patient came to a hospital emergency room. She stated that she had eaten ham the day before, and in the past had had a "reaction" to this food. A history of recurrent headaches was given, and the patient reported a recent diagnosis of hypertension. Findings on neurological examination were normal, including results of funduscopic examination, though the presence or absence of nuchal rigidity was not recorded. Blood pressure ranged from 170/100 to 210/120 mm of mercury. Antiemetics and mild analgesics were given and the patient

was sent home with a diagnosis of acute gastroenteritis.

Four days later the patient was admitted to hospital after presenting to another physician with left hemiparesis and mild confusion. Blood pressure at that time was 160/108 mm of mercury. Results of funduscopic examination were normal and there was no nuchal rigidity.

On lumbar puncture, bloody cerebrospinal fluid with xanthochromic supernatant was noted. By the next day, lethargy and hemiplegia with a tendency toward decerebrate posturing on the left were present.

Findings on cerebral angiography indicated a large right internal carotid aneurysm, along with severe vasospasm of the internal carotid, middle cerebral and anterior cerebral arteries (Figure 4). At that point, the patient was not a candidate for surgical operation. Large doses of dexamethasone were given, but the patient continued to deteriorate and died 24 hours later.

Comment

This patient's case represents an extremely difficult problem in the management of subarachnoid hemorrhage. Cerebral vasospasm and progressive neurological deterioration following subarachnoid hemorrhage in a hypertensive patient is very frequently a lethal chain of events. Attempts to treat the hypertension by lowering the blood pressure may accentuate the cerebral ischemia. Attempts to increase cerebral perfusion by elevating the blood pressure are liable to cause massive hemorrhage from the aneurysm.

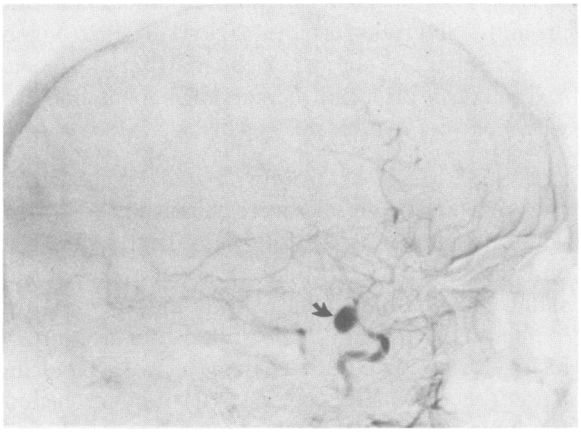

Figure 4.—(Case 5) Headache, nausea and vomiting in a 37-year-old woman was initially diagnosed as gastroenteritis. On angiography five days after onset of the symptoms, a large right internal carotid artery aneurysm (arrow) was seen. Severe vasospasm had developed by that time and no effective therapy was available. The patient died.

Diagnosis of the subarachnoid hemorrhage at the time of the patient's first presentation to the emergency room would have provided the only chance for survival. Obliteration of the aneurysm a day or two after the hemorrhage would have allowed treatment of the vasospasm when its effects became severe by attempting to increase cerebral perfusion either by further elevating the blood pressure with vasopressors or by carrying out a microsurgical vascular bypass.

Discussion

Pakarinen has shown that 32 percent of patients with ruptured intracranial aneurysms die within 24 hours of the major initial hemorrhage, and 35 percent die within three days.[17] However, in many patients before a major hemorrhage there are warning signs indicating expansion of the aneurysm or minor leakage from it.[8,18,19] Patients can be protected from the high initial mortality of a major hemorrhage if these warning signs are recognized and the aneurysm treated while the patient is still neurologically intact.

Okawara analyzed 112 patients with ruptured aneurysms and showed that in 48.2 percent there were warning signs before the major hemorrhage.[18] The incidence of warning signs in patients harboring internal carotid artery aneurysms at the origin of the posterior communicating artery was 69.2 percent. The average interval between the onset of warning signs indicating minor leakage of blood into the subarachnoid space and a major subarachnoid hemorrhage was 10.4 days.

Waga, Ohtsubo and Handa analyzed the clinical histories of 192 patients with intradural intracranial aneurysms.[19] In 49 percent of the 98 men and in 69.1 percent of the 94 women there were warning signs before a major hemorrhage. Warning signs occurred in 56.5 percent of patients with anterior cerebral aneurysms, in 48.8 percent of those with middle cerebral aneurysms and in 68.8 percent of those with intradural internal carotid aneurysms.

King and Saba found that in 106 (64 percent) of 165 patients with ruptured berry aneurysms, there were symptoms and signs forewarning a major subarachnoid hemorrhage, and a third of these patients had been treated for erroneously diagnosed illness for more than a week before subarachnoid hemorrhage was considered as a diagnostic possibility.[8]

In these studies, warning signs included sudden unusual headache, nausea and vomiting, transient loss of consciousness and transient visual disturbances. Expansion of an unruptured posterior communicating artery aneurysm can cause oculomotor nerve paralysis, resulting in various degrees of pupillary dilatation, ptosis and turning of the eye to the characteristic "down and out" position of third cranial nerve palsy.

TABLE 1.—*Misinterpretation of Signs and Symptoms in Subarachnoid Hemorrhage*

Headache
Migraine
Sinusitis
Tension headache
Hypertensive headache
Subdural hematoma
Nuchal rigidity
Cervical strain
Ruptured cervical disc
Meningitis
Nausea and vomiting
Acute gastroenteritis or food poisoning
Peptic ulcer disease
Acute appendicitis (with tenderness of the abdominal wall)
Anterior myocardial infarct (with epigastric or anterior thoracic discomfort)
Confusion, lethargy or personality change
Drug overdose
Alcoholic intoxication
Acute psychosis
Hemiparesis or other focal neurological deficit
Cerebral infarction from thrombosis or embolism
Spontaneous intracerebral hemorrhage

TABLE 2.—*Initial Evaluation of Suspected Subarachnoid Hemorrhage*

History and general physical examination
Look for nuchal rigidity and elevated blood pressure.
Neurological examination
Look for signs of oculomotor nerve palsy, hemiparesis or other focal neurological signs.
Funduscopic examination
Look for retinal or preretinal hemorrhage.
X-ray studies of the skull
Look for intracranial calcification, an enlarged sella turcica, thinning of the dorsum sellae, or other signs of an intracranial mass lesion or elevated intracranial pressure.
Lumbar puncture
(Withhold lumbar puncture if retinal hemorrhages, papilledema or other signs of increased intracranial pressure are present.)
Look for blood or xanthochromia in the cerebrospinal fluid (CSF). A traumatic tap is distinguished by clearing of blood by the third tube and by the absence of xanthochromia in the centrifuged CSF.
Cerebral angiography
This should be done if subarachnoid hemorrhage is confirmed.

The initial symptoms in Case 4, above, fall into the category of warning signs. The initial symptoms in the other four patients reflect what this author would term major subarachnoid hemorrhage. As the case reports show, the symptoms and signs of subarachnoid hemorrhage can be misinterpreted in many ways (Table 1). The headache can suggest the presence of migraine, sinusitis, tension headaches, hypertensive headache (see Case 1) or subdural hematoma (see Case 2). The precipitous nature of the headache associated with subarachnoid hemorrhage is usually characteristic. Migraine usually has its onset before the age of 30 years and is not an isolated event. A single headache occurring suddenly in a middle-aged person is not very likely migraine. If there is any question concerning the diagnosis of migraine headache, a lumbar puncture should be done to rule out subarachnoid hemorrhage.

The nuchal rigidity of subarachnoid hemorrhage can suggest cervical strain (see Cases 3 and 4) or ruptured cervical disc. The sudden onset, the absence of a specific precipitating traumatic event and the usual association of the rigidity with occipital or generalized headache should make the examiner suspect that he is not dealing with a cervical syndrome. The precipitous onset should also differentiate the syndrome from meningitis, though a misdiagnosis of meningitis will at least lead to a lumbar puncture and the true nature of the disease process will be defined.

The nausea and vomiting associated with subarachnoid hemorrhage may mask the importance of the accompanying headache, and be misinterpeted as gastroenteritis (see Cases 1, 3 and 5). Prolonged vomiting may result in tenderness of the abdominal wall, thus leading to the diagnosis of an acute abdomen. If epigastric discomfort is prominent, occult anterior myocardial infarction may be considered (see Case 3).

Subarachnoid hemorrhage at times leads to lethargy, confusion or personality change misinterpreted as drug or alcoholic intoxication or as psychiatric disease (see Case 3). The absence of a history of drug overdose or alcoholic indiscretion along with the presence of a headache should help in narrowing the differential diagnosis.

Subarachnoid hemorrhage may be associated

with hemiparesis or other focal neurological deficit as the result of cerebral vasospasm or as the result of hemorrhage from the aneurysm into the cerebral substance or into the subdural space. Since both cerebral infarction from thrombosis or embolism and spontaneous intracerebral hemorrhage are often associated with blood in the subarachnoid space, angiography may at times be the only way in which to rule out a ruptured intracranial aneurysm as the cause of the patient's neurological compromise.

A patient in whom cranial, occipital or cervical pain has developed suddenly, especially if associated with stiff neck, syncope, confusion, personality change, hemiparesis, sensory changes, visual disturbances or seizures, should have careful neurological and funduscopic examinations, along with a lumbar puncture (Table 2). The lumbar puncture should be withheld in favor of angiography or computerized tomographic studies ("EMI" scanning) if the patient has retinal hemorrhages, papilledema or other signs of increased intracranial pressure. In all other patients in whom subarachnoid hemorrhage seems a diagnostic possibility, lumbar puncture should be done without hesitation. The indications for lumbar puncture and the techniques of the tap have been thoroughly reviewed recently.[20] The tap can be done in the office or emergency room with a No. 22 spinal needle. If there is no blood in the cerebrospinal fluid, and if there is no other evidence of a disorder requiring admission to hospital, the patient can be released after an hour or two of rest and observation.

If blood is present in the cerebrospinal fluid, a traumatic tap is usually distinguished by clearing of the fluid by the time the third tube is collected. The supernatant of the centrifuged specimen will usually, but not always, be xanthochromic if true subarachnoid hemorrhage is present.

If subarachnoid hemorrhage is confirmed, then cerebral angiography should be carried out promptly, preferably within 24 hours of the patient's admission, so that operative intervention or other appropriate therapy can be planned and instituted at the optimal time. If the patient is comatose or deteriorating, then angiography should be done as an emergency procedure to rule out an intracerebral or subdural hematoma possibly amenable to immediate evacuation.

Treatment of ruptured intracranial aneurysms by nothing more than bedrest and hopeful waiting is becoming passé. Whether an aneurysm is treated by microsurgical obliteration, antifibrinolytic therapy, stereotactic obliteration, a nonsurgical hypotensive regimen or endovascular obliteration by selective catheterization of the intracranial arteries, therapy must be planned and instituted as soon as possible—preferably before a major subarachnoid hemorrhage has occurred.

Our diagnostic goals should be prompt recognition of patients in whom there are symptoms and signs which reflect impending aneurysmal rupture and immediate recognition of patients in whom major subarachnoid hemorrhage has occurred. It appears that in a significant percentage of patients these goals are not being attained because subarachnoid hemorrhage is often an insidious process and may initially masquerade in many guises. The physician may be lulled into a false sense of diagnostic security until the patient deteriorates precipitously and passes beyond therapeutic reach.

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